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The neuroendocrine effects of the TASER X26®: A brief report

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ABSTRACT

Introduction: Law enforcement officers use conducted electrical weapons (CEW) such as the TASER X26® to control violently resistive subjects. There are no studies in the medical literature examining the effects of these weapons on the human stress response. This is the first study to compare the human stress response to conducted electrical weapons, oleoresin capsicum (O.C.), a cold-water tank immersion, and a defensive tactics drill.

Methods: Subjects were randomized to one of the four interventions studied. Subjects received either a 5-s exposure from the TASER X26 CEW with the probes fired into the back from 7 ft, a 5-s spray of O.C., a skin and mucous membrane irritant, to the eyes, a 45-s exposure of the hand and forearm in a 0 °C cold water tank, or a 1-min defensive tactics drill.

Results: Alpha-amylase had the greatest increase from baseline at 10–15 min with the defensive tactics drill. Cortisol had the greatest increase at 15–20 min with O.C. Cortisol remained most elevated at 40–60 min in the defensive tactics drill group.

Conclusions: Our preliminary data suggests that physical exertion during custodial arrest may be most activating of the human stress response, particularly the sympathetic–adrenal–medulla axis. This may suggest that techniques to limit the duration of this exertion may be the safest means to apprehend subjects, particularly those at high-risk for in-custody death. Conducted electrical weapons were not more activating of the human stress response than other uses of force.

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1. Introduction

The TASER X26® is a handheld conducted electrical weapon (CEW), powered by two 3 V batteries, that induces neuromuscular incapacitation and pain by the application of a small electrical current. The electrical current stimulates both afferent sensory neurons causing pain, and efferent motor neurons causing involuntary skeletal muscle contraction. Law enforcement officers utilize the weapon to obtain physical control of violently resistive subjects. There has been controversy in the lay press with regard to the use of these weapons and sudden in-custody death. Claims have been made that these weapons have been responsible for greater than 150 such deaths [1]. There has been speculation that exposure to the discharge of a conducted electrical weapon may induce neuroendocrine effects which might predispose subjects to sudden death. Currently, there are no studies of the neuroendocrine effects of these weapons. This brief report is an attempt to examine the neuroendocrine effects of the TASER X26 as compared to other standard law enforcement uses of force.

2. Methods

This was a prospective study of adult human volunteers. Volunteers were recruited for the study from law enforcement training courses. The study was conducted either on the premises of the CEW manufacturer or at training conference sites. All interventions in the study were usual aspects of their law enforcement training except for the cold-water tank immersion described below. Volunteers were provided a TASER X26 CEW as compensation for their participation. The study was approved by the institutional review board (IRB) at Hennepin County Medical Center (Minneapolis, MN). All subjects provided informed consent prior to enrolment.

All subjects completed a medical screening questionnaire that was reviewed by an investigator prior to testing. There were no specific exclusion criteria other than all participants had to be at full duty status with their departments (this automatically excluded subjects with recent orthopedic injury or surgery). The investigator

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Table 1
Demographics.

	Subjects (male/female)	Age	BMI
Cold-water tank	16 (14/2)	42.9 ± 9.2 (28–61)	28.5 ± 4.3 (19.7–38.4)
CEW	16 (16/0)	34.5 ± 7.4 (22–49)	28.9 ± 4.2 (22.6–37.3)
O.C.	10 (8/2)	28.4 ± 7.4 (19–38)	29.9 ± 5.0 (23.1–38.8)
Defensive tactics drill	10 (10/0)	44.6 ± 2.3 (35–59)	29.9 ± 5.2 (21.8–38.7)

Table 2
Neuroendocrine salivary biomarker baseline values.

		Cold-water tank	O.C.	Defensive tactics	CEW
Amylase (U/mL)	Median	158	59.0	264	132.3
	IQR	73.8–188.6	23.1–118.2	152.2–344.7	64.4–202.7
	Range	14.9–255.0	14.9 to	69.2–805.0	29.7–287.8
Cortisol (mcg/dL)	Median	0.17	0.50	0.28	0.47
	IQR	0.14–0.30	0.36–0.55	0.21–0.30	0.33–0.78
	Range	0.07–1.13	0.16–0.39	0.33–0.78	0.15–1.55

could exclude subjects if he felt that participation would place the subject at substantial risk of injury, particularly musculo-skeletal injury due to a pre-existing condition. An investigator observed all subjects during the testing, and had the ability to halt the study for any reason.

The primary measured outcome was salivary alpha-amylase and salivary cortisol. Recent literature has shown that salivary markers may be able to be used to quantitatively measure the stress response in subjects exposed to stress [2–5]. The advantage of salivary markers is eliminating the stress of a needle stick to draw serum measures. Salivary alpha-amylase has been suggested as a measure of the sympathetic–adrenal–medulla (SAM) axis. There is some debate as to whether this measure reflects catecholamine levels or is a more general measure of sympathetic tone [6–9]. Salivary cortisol has been suggested as a measure of the hypothalamus–pituitary–adrenal (HPA) axis. According to Salimetrics Inc. these measures peak 10–20 min after the presentation of the noxious stimulus, with alpha-amylase peaking at about 10 min, and cortisol at about 20 min.

Subjects on each day were a convenience sample from a concurrent training course. Subjects were randomized to one of the four interventions studied. Subjects received either a 5-s exposure from the TASER X26 CEW with the probes fired into the back from 7 ft, a 5-s spray of oleoresin capsicum (O.C.), a skin and mucous membrane irritant, to the eyes, a 45-s exposure of the hand and forearm in a 0-°C cold water tank, or a 1-min defensive tactics drill. The O.C. used was Def Tac 10% pepper foam. Subjects had to face forward with eyes open and mouths closed. No instructions were given as to breathing. The subjects exposed to the O.C. were allowed to use towels and water rinses immediately after exposure. The defensive tactics drill consisted of 1 min of the subject preventing multiple defensive tactics instructors from trying to remove a simulated handgun from his holster on his duty belt while in the supine position on a training mat. The cold-water tank was used since it has been established as a standard painful stimulus in the neuropsychiatric literature (referred to commonly as the cold pressor task) [10–12].

Subjects had salivary samples collected by passive drool through a straw 10–15 min before the exposure, and at 10–20 min after the exposure, and 40–60 min after the exposure. Salivary samples were packed in dry ice and shipped overnight to Salimetrics Inc. (State College, PA) for analysis for quantitative measures of alpha-amylase and cortisol.

Data were entered in an Excel® (Microsoft Corp., Redmond, WA) spreadsheet and analyzed using STATA 10.0 (Stata Corp., College Station, TX). Descriptive statistics were used as appropriate. Salivary amylase and cortisol values for each intervention were compared between time-points using Wilcoxon sign rank tests. Values at each time-point were compared by intervention using Wilcoxon rank sum tests. In order to have a 90% probability of detecting a difference between the TASER X26 group and the other interventions with a power of 80% and a significance of 0.05, 10 subjects were needed in each group.

3. Results

A total of 53 subjects were enrolled. Only one subject was excluded secondary to a recent knee injury. There were 16 subjects in the cold-water tank group, 16 subjects in the CEW group, 10 in the O.C. group, and 10 in the defensive tactics drill group. There were no adverse outcomes reported. The demographics for each group are presented in Table 1.

The results are presented in Tables 2 and 3, and, graphically, in Figs. 1 and 2. Alpha-amylase had the greatest increase from baseline at 10–15 min with the defensive tactics drill. Cortisol had the greatest increase at 15–20 min with O.C. Cortisol remained most elevated at 40–60 min in the defensive tactics drill group.

4. Discussion

The acute stress response in humans is a neuroendocrine cascade initiated by the hypothalamus. The cascade has two components: the sympathetic–adrenal–medulla axis, and the hypothalamic–pituitary–adrenal axis. The SAM axis is responsible for the release of catecholamines, primarily epinephrine, from the adrenal medulla chromaffin cells. These cells contain a pool of catecholamines that are available for immediate release. This axis is colloquially referred to as the "fight or flight" response. The HPA axis is responsible for the release of ACTH and β-endorphins. ACTH causes the adrenal cortex to produce glucocorticoids (e.g., cortisol) and mineralocorticoids. β-endorphins modulate pain perception. Cortisol induces an enzyme in the adrenal medulla that is the rate-limiting step in the conversion of norephinephrine to epinephrine. Cortisol also induces gluconeogenesis. The mineralocorticoids increase blood volume. The effects of this axis are complex and include changes which allow humans to continue the "fight or flight" as well as changes to limit the damage from the stress response itself [13].

Epinephrine release is the key end-result of the neuroendocrine cascade. It produces a number of adaptive physiologic changes in response to stress, including positive chronotropic and inotropic

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Table 3
Neuroendocrine salivary biomarker change after stressor.

	10–15 min	*P*-value (Wilcoxon sign rank)	40–60 min	*P*-value (Wilcoxon sign rank)
Amylase (U/mL)				
Cold-water tank				
Median	−35.3	0.46	2.6	0.71
IQR	−86.8 to 55.8		−35.3 to 80.8	
Range	−185.6 to 125.2		−109.8 to 250.8	
O.C.				
Median	37.4	0.01	8.4	0.03
IQR	17.7 to 70.7		2.6 to 29.4	
Range	−255.0 to 154.3		−5.1 to 03.2	
Defensive tactics				
Median	63.8	0.51	−84.5	0.09
IQR	−70.2 to 209.6		−122.7 to −9.2	
Range	−267.9-514.3		−433.3 to 197.8	
CEW				
Median	−4.5	0.15	−20.1	0.21
IQR	−77.5 to 11.2		−53.3 to −5.2	
Range	−197.6 to 60.1		−122.2 to 77.2	
	15–20 min	*P*-value (Wilcoxon sign rank	40–60 min	*P*-value (Wilcoxon sign rank)
Cortisol (mcg/dL)				
Cold-water tank				
Median	0.07	0.42	0.04	0.96
IQR	−0.001 to 0.15		−0.06 to 0.125	
Range	−0.45 to 0.43		−0.45 to 0.43	
O.C.				
Median	0.50	0.01	0.01	0.96
IQR	0.13–0.58		−0.20 to 0.19	
Range	−0.08 to 0.90		−0.33 to 0.89	
Defensive tactics				
Median	0.25	0.007	0.47	0.005
IQR	0.18–0.48		0.43–0.62	
Range	−0.04 to 0.55		0.06–1.36	
CEW				
Median	0.38	0.002	0.32	0.011
IQR	0.15–0.46		0.003 to −0.50	
Range	−0.21 to 0.58		−0.30 to 0.93	

cardiac effects, increased systemic vascular resistance, increased arterial blood pressure, increased metabolism, and increased thermogenesis. However, it can also produce maladaptive physiologic changes including myocardial ischemia, cardiac dysrythmias, reflex bradycardia (which can cause asystole), pulmonary edema, lactic acidosis, and hyperthermia [13].

It has been hypothesized that these maladaptive physiologic changes may be contributory to some in-custody deaths. This may be especially the case when the acute stress response is potentiated by the use of cocaine, amphetamines, phencyclidine (PCP) or other stimulants, or when psychiatric illness or paranoid drug-intoxicated states induce a greater perceived threat from the police contact and therefore a greater acute stress response, or when the subject is in a state of excited delirium.

Karch and Stephens described histopathological "contraction bands" in subjects who died of excited delirium [14]. Contraction band necrosis is a unique form of cardiac myocyte injury that has been related to catecholamines. They have been observed in catecholamine excess states such as pheochromocytoma and subarachnoid hemorrhage and at autopsy in persons who died terrifying deaths [15]. Additionally, lactic acidosis and hyperthermia, maladaptive changes from epinephrine, have been found in many cases of in-custody death [14,16].

The phenomenon of "capture shock" has been well described in the veterinary literature. It has been observed in many animal species and is related to the stress of capture [17–20]. The changes described in this phenomenon mimic the changes seen in many in-custody deaths, particularly those from excited delirium. There are case reports of medically supervised restraint deaths in humans in the geriatric and psychiatric literature [13]. In humans, myocardial stunning has been observed after severe emotional stress. The phenomenon has been referred to as "tako-tsubo" cardiomyopathy in Japan, and represents about 1% of admissions for myocardial infarction in Japan. In a series by Sharkey et al. in Minneapolis, MN, there was about one case per month [21]. Wittstein et al. studied 19 previously healthy patients with myocardial stunning after sudden emotional stress. These patients presented with chest pain, pulmonary edema, and shock. The median ejection fraction was 0.20. Catecholamine levels in these patients were 2–3 times those of Killip class III myocardial infarction patients and 7–34 times those of published normal values. Wittstein et al. proposed several mechanisms for the association of sympathetic stimulation and myocardial stunning: (1) epicardial coronary artery spasm, (2) microvascular spasm, and (3) direct myocyte injury [15]. There is speculation that CEW exposure may contribute to the in-custody death phenomenon by inducing similar stress mechanisms.

There are also in-custody death theories about the delayed clearance of catecholamines from the blood after the cessation of the stressor. Based on the work of Dimsdale et al. and others, Di Maio proposed that persistent circulating levels of catecholamines create a "period of peril" after the cessation of struggle. Catecholamines cause potassium to enter cells. This is thought to be

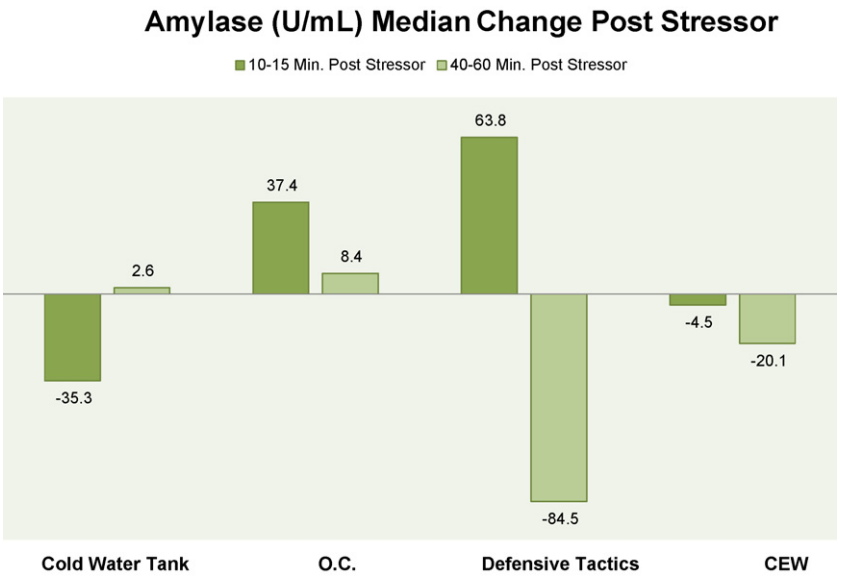

Fig. 1. Amylase (U/mL) median change post-stressor.

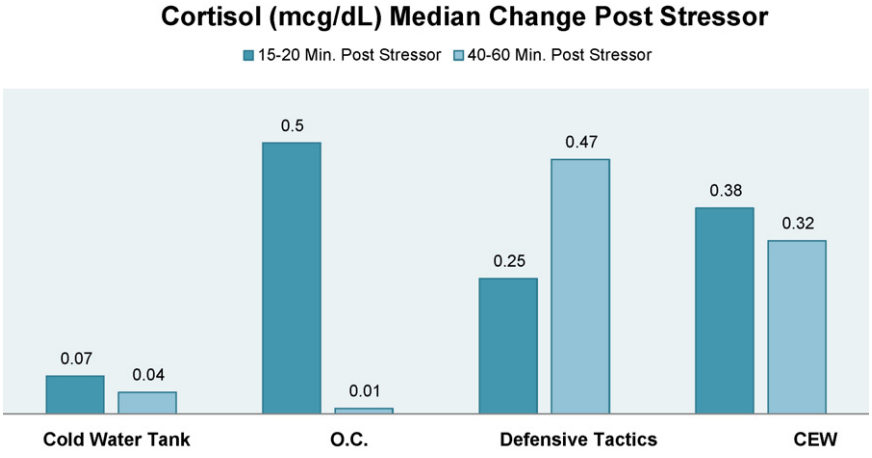

Fig. 2. Cortisol (mcg/dL) median change post-stressor.

protective during the "fight or flight" response as potassium leaks out of exerting muscle tissue. However, once the exertion ceases, the persistent circulating catecholamines can cause a temporary hypokalemia [22,23]. Hagberg et al. found that norepinephrine increased during the first minute post-exercise, and then decreased with a half-life of 2.8 min between 1 and 11 min after exercise [24]. Young et al. found a mean rate of decline of potassium after exercise to be −0.54 mequiv./(L min) [25]. Coplan et al. found that potassium shifts were more exaggerated with exercise above the lactate threshold than below the lactate threshold [26]. Hypokalemia can lead to cardiac dysrhythmias as well as respiratory arrest independent from cardiac arrest. This has been proposed as a possible mechanism for the sudden death seen after the cessation of struggle in many in-custody deaths [22]. In contrast, in a swine study by Jauchem et al., potassium was significantly elevated after prolonged CEW discharge [27]. This elevation could be attributed to the severe combined respiratory and metabolic acidosis in these animals. In human studies of 5–15 s CEW discharges, no clinically important changes in potassium occurred with the discharges [28,29].

Fletcher at al. noted that the post-exercise period can be dangerous because of the peripheral arteriolar dilation associated with exercise. The vasodilation coupled with a sudden decrease in venous return from the termination of muscular activity can reduce cardiac output suddenly and reduce coronary artery perfusion while the heart rate is still elevated [30]. Rywik and Zink found that asymptomatic ischemic ST-segment changes that begin after the cessation of exercise have a similar adverse prognostic significance to ischemic ST segment changes occurring during exercise. They proposed that the mechanism for these ST-segment changes during recovery might be the augmentation of catecholamines in the early post-exercise period [31]. In addition, the persistent circulating catecholamines can continue to exert other maladaptive effects such as increased metabolism and hyperthermia. Many of these changes may not be evident on autopsy, and may explain the autopsy-negative death in some cases [13].

Catecholamines can also cause asymptomatic disease states to become life-threatening problems. Mild-moderate asymptomatic atherosclerotic disease can cause ischemia and arrhythmias in the presence of high circulating catecholamines. Animal studies have shown that hypertensive animals have greater changes in heart rate, blood pressure, and temperature than normotensive animals in response to catecholamines [13]. The long-QT syndrome is characterized by exercise or stress-induced syncope or sudden

death [32]. One of the initial descriptions in the medical literature of Jervell Lange-Nielson syndrome, a congenital long QT syndrome, was a case report of a child who died suddenly while being admonished [13]. Lampert et al. have hypothesized catecholamine induced repolarization changes may be the cause of stress-related arrhythmias [33].

In addition, catecholamines in the right clinical milieu might be a cause of sudden death. "Huffing" aerosolized paint can cause sudden death. The mechanism is attributed to the sensitization of the myocardium from the aerosolized paint, and then sudden surprise (being caught) leading to a catecholamine surge and fatal arrhythmia [34]. Likewise, elevated catecholamines in the presence of cocaine or other sympathomimetic drugs can increase the toxicity of the drugs [35].

Lastly, there are theories of sudden collapse from adrenergic failure. Transient adrenal insufficiency in critical illness has been described in the critical care literature. The mechanisms are complex and may involve a systemic inflammatory response suppression of the hypothalamus or a down-regulation of adrenergic receptors leading to vascular hypo-responsiveness and myocardial depression [36]. Cocaine washout syndrome has also been described in case reports and is characterized by somnolence and possibly mild hypotension and mild bradycardia. The mechanism has been proposed as catecholamine depletion [37].

Wittstein et al. evaluated 19 patients who had myocardial stunning after sudden emotional stress. All of the patients were symptomatic with chest pain, dyspnea, or both. Diffuse T-wave inversion and a prolonged QT interval were seen in most of the subjects. In 17 of the 19, there was a mild elevation in troponin. All of the subjects had severe left ventricular dysfunction [15]. In the study by Ho et al., none of the subjects had subjective complaints of chest pain or shortness of breath. None of the subjects had electrocardiogram changes. One subject had a mild elevation in troponin, but had a complete in-hospital cardiac evaluation and no abnormalities were found [28]. This seems to suggest that CEWs are not producing the changes seen in myocardially stunned patients.

In this study, the TASER X26 CEW was compared to other uses of force or an established painful stimulus. The defensive tactics drill resulted in the greatest change in salivary alpha-amylase at 10–15 min with a change of 63.8 U/mL. O.C. was next with a change of 37.4. The CEW and cold-water tank immersion did not appear particularly activating of the sympathetic stress response. O.C. had the greatest change in salivary cortisol at 15–20 min with a change of 0.5 mcg/dL. The CEW was next with a change of 0.38, and the defensive tactics drill after that with a change of 0.25. The defensive tactics drill had the greatest delayed change from baseline in cortisol with a change of 0.47. The cold-water tank immersion did not appear particularly activating of the HPA stress response. While statistical significance is limited by this data, it tends to suggest that exertion and O.C. may have the most important influence on these markers of stress when compared to the cold-water immersion tank or the TASER CEW.

While no other data exists in the literature comparing a CEW discharge to exertion, there is a study that compared symptom-limited hand grip, the cold-pressor task (equivalent to the cold tank in this study), and symptom-limited supine bicycle exercise in relation to catecholamine responses. In this study, catecholamine levels were 3–6 times greater in the symptom-limited supine bicycle exercise group than the other two groups. This suggests that exertion is a much more potent stimulant for catecholamines than pain-related stimuli [38].

In a study by Han et al. in rats, it was found that cocaine combined with exercise increased epinephrine, norepinephrine, and lactate 2–5 times greater than either exercise or cocaine alone, and 11–35 times greater than rest with no cocaine [39]. This study also points to the significant activation of the stress cascade with exertion, but more, demonstrates the much greater effect when exertion is compounded with acute drug use. Other animal studies have shown a relationship between restraint stress and sensitization to drugs of abuse. In a study by Pacchioni et al., it was found that a single restraint exposure was sufficient to cause a significantly increased release of dopamine and locomotor activity to an amphetamine challenge in rats [40]. A study by Pudiak et al. demonstrated a significantly higher mortality with cocaine and restraint in rats compared to cocaine alone. These authors concluded that minimizing the stress response may be important in cocaine toxicity [41]. In a study by Pacak and Palkovits, rats were subjected to one of five stressors: cold, hypoglycemia, hemorrhage, pain (formulin injection into a limb), and immobilization. The authors found that immobilization increased ACTH the most, followed by hypoglycemia. Cold stress increased norepinephrine the most, followed by immobilization. Hypoglycemia increased epinephrine the most, followed by immobilization. The immobilization stress was consistently one of the highest stressors by these measures [42]. A study by Sun et al. showed that restraint stress decreased ventricular electric stability making it more sensitive to arrhythmogenic drugs [43]. Sanchez et al. demonstrated several biomarker indications of injury, including heart muscle injury, with restraint and confrontational stress in a murine model [44]. These studies may provide insight into causation in many of these sudden in-custody death cases.

Law enforcement officers are confronted with limited options to control violent subjects. In this study, we attempted a preliminary investigation into the stress response induced by several of these options (with the weapon retention drill as a surrogate for physical arrest). This is important since some have speculated that conducted electrical weapons may be contributory in these deaths by a stress-related mechanism. Our preliminary data suggests that physical arrest (exertion) may be one of the most activating stressors of the human stress response, particularly with regard to the sympathetic response, and might suggest that techniques to limit the duration of this exertion may be the safest means to apprehend subjects, particularly those at high-risk for in-custody death.

The mechanisms in the sudden, unexpected in-custody death phenomenon have not been elucidated. It is likely that many pathological processes are involved. It is likely that the stress response has some contribution. There are other disease processes in which the stress response is known to be or hypothesized to be contributory in sudden death such as certain genetic arrhythmia conditions (e.g., long QT syndrome), sudden unexplained death in epilepsy (SUDEP), huffing deaths, etc. The in-custody death phenomenon is likely a "perfect storm" of extreme physiologic conditions including an exaggerated stress response, potentiated by drugs of abuse, physical exertion and restraint, with hyperthermia, acidosis, electrolyte changes, autonomic dysregulation, and exacerbation and/or unmasking of prior health problems. Chronic drug abuse may have a "kindling" effect that creates the neuro-physiologic changes that put these processes in motion.

5. Limitations

There are several limitations in this study. First, the volunteers were recruited from law enforcement training courses. The subjects were under variable baseline stress from these courses depending upon the nature of the training. Second, due to logistics, it was difficult to control the subject behavior prior to entry in the testing room. Salimetrics Inc. had recommendations about oral

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intake prior to testing which was difficult to control in our environment. Third, it is unclear whether the fact that O.C. is a primary skin and mucus membrane irritant caused an alteration in the salivary measures. Fourth, the numbers in this study were small. Fifth, although we attempted to keep the nature of the testing unknown to the subjects until immediately before the intervention, this was practically difficult to achieve. Lastly, several of the defensive tactics drill subjects were defensive tactics instructors and had participated in similar drills previously. In addition, almost all law enforcement academies require O.C. exposure to graduate so all participants had prior exposure to O.C. This prior experience could have made the interventions less stressful. A least one defensive tactics drill subject described the drill as "fun." This less simulates the "capture stress" of an arrest than the study investigators had expected. By contrast, most of the CEW subjects had never had a prior exposure. It is possible the difference in the stress response between the CEW and the defensive tactics drill and O.C. would have been greater if subjects had not had prior experience with these interventions.

6. Conclusions

Our preliminary data suggests that physical exertion during custodial arrest may be most activating of the human stress response, particularly the sympathetic–adrenal–medulla axis. This suggests that tools and techniques to limit the duration of this exertion may be the safest means to apprehend subjects, particularly those at high-risk for in-custody death. Conducted electrical weapons were not more activating of the human stress response than other uses of force in our study. We recommend further study to determine the mechanisms involved in the in-custody death phenomenon.

Conflicts of interest

Dr. Dawes and Dr. Ho would like to declare the following conflicts: external medical consultants to TASER International, stockholders.

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